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THE USE OF QUINIDIN IN THE TREATMENT OF HEART DISORDERS

Since Wenckebach in 1914 reported the specific effect of quinin upon auricular fibrillation and Frey in 1918 showed the more effective action of quinidin sulphate, the latter drug has been given a fair trial throughout the medical world in the treatment of multiple premature contractions (extrasystoles), paroxysmal tachycardia, auricular fibrillation and flutter. More recently this drug has been recommended for patients suffering from coronary thrombosis with premature beats or ventricular tachycardia, in the hope of lessening myocardial irritability and thus arresting ventricular tachycardia and perhaps preventing ventricular fibrillation, which may be the cause of sudden death in such cases.

Unfortunately many physicians have gained the impression that quinidin sulphate is similar to digitalis in its action. This, of course, is far from the truth as quinidin has a depressant effect upon the myocardium. In fact this drug must be given with great caution, since cases of sudden death have been reported in which quinidin has been suspected of producing "ventricular tachycardia and ventricular fibrillation"; "sudden standstill of the heart"; "fatal toxic effects upon the myocardium"; "respiratory paralysis"; and "death due to embolism from an intra-auricular thrombus."

Apparently in cardiovascular disease the only possible benefit to be derived from quindin is through preventing or arresting the various forms of arhythmias and paroxysmal tachycardias. Hence this drug is absolutely contra-indicated in the presence of a normal sinus rhythm with congestive circulatory failure, so-called cardiac decompensation. In fact when it is given for some form of arhythmia, the circulation is generally affected adversely while this transition is being produced. In other words, the patient is often made worse temporarily in the attempt to improve the condition. The drug should therefore not be given for a long time, unless the desired result has been obtained. In most cases not more than a few days or a week should be used in deciding whether quinidin will regularize the heart or not.

Electrocardiographic evidence of auricular standstill associated with quinidin therapy of auricular fibrillation, has been published; and standstill of the whole heart due to depression of ventricular as well as the auricular pacemaker, has been suspected as the cause of sudden death in quinidin therapy.

The method of administering quinidin sulphate has become fairly well established. It is given in tablets, powders or capsules of three grains (0.2 gram). If a preliminary dose fails to develop an individual idiosyncrasy to the drug, it is usually repeated at intervals of four hours during the day in ambulatory cases with extrasystoles or paroxysmal tachycardias, and this dosage may be continued indefinitely if untoward symptoms do not develop. As quinidin sulphate is rapidly eliminated, the same dose may be given every second hour, day and night, but these patients should be kept constantly in bed under careful supervision and, if possible, where frequent electrocardiographic observations may be made. If the objective is not obtained in twenty-four or forty-eight hours, the dosage may be doubled, six grains (0.4 gram) every second or fourth hour and continued for seven to ten days, if symptoms of cinchonism, such as marked tinnitus, deafness, urticaria, nausea, vomiting or diarrhea do not develop. There are rare occasions when the arhythmia itself is the cause of the sudden serious complication and then the doses may be increased much more rapidly.

Premature Contractions (Extrasystoles)

If the removal of the toxic factors usually suspected of producing this arhythmia, in the absence of organic heart disease, has failed to eliminate it and if the premature contractions are noticed by the patient, thus warranting further treatment, quinidin sulphate may be successful in clearing up these premature beats in about 5% of the cases. The same results may be expected in cases of rheumatic or hypertensive arteriosclerotic cardiovascular disease complicated by premature contractions. Unfortunately a persistent, debilitating diarrhea often necessitates stopping the drug. At times, however, even though the drug must be stopped, the arhythmia does not return.

Paroxysmal Auricular Fibrillation and Paroxysmal Tachycardias

Quinidin sulphate has reduced the number of such attacks in certain patients and has eradicated

them entirely in others. It would seem, however, that full digitalization with a daily maintenance dosage of this drug, has proved more successful in terminating and preventing paroxysmal auricular fibrillation and paroxysmal tachycardias than has quinidin. The termination of a few cases of paroxysmal auricular tachycardia by intravenous injection of three grain (0.2 gram) of quinidin has been reported. Quinidin sulphate administered orally has terminated attacks of ventricular tachycardia both paroxysmal and when associated with coronary occlusion.

Chronic or Established Auricular Fibrillation

In the vast majority of such cases, the best procedure seems to be full digitalization and a daily maintenance dose for the rest of the patient's life. In a few cases in which apparently no true myocardial damage is present and with a restoration of sinus rhythm the heart can be considered normal, quinidin therapy is indicated. There are still some authorities who are more enthusiastic about the value of quinidin and advise its use for auricular fibrillation even in the presence of organic myocardial or valvular disease. It is wise to digitalize most of these patients before starting quinidin therapy. In all cases of chronic auricular fibrillation, normal sinus rhythm can be obtained in from 60% to 80% of cases and in most can be maintained for weeks or months. In a few of these cases, after a restoration of normal sinus rhythm, it has been maintained, with or without constant quinidin sulphate therapy, for a period of from five to ten years.

In auricular fibrillation associated with hyperthyroidism, if normal sinus rhythm does not return spontaneously seven to ten days after partial thyroidectomy, it can be restored with quinidin sulphate in practically 100% of cases, if thyrotoxicoses has been the sole etiological factor and the hyperthyroid state has been cured by the operation.

Chronic Auricular Flutter

If after digitalization, auricular flutter reverts to auricular fibrillation, a final reversion to normal sinus rhythm may be hastened by quinidin therapy. If in auricular flutter digitalization proves unavailing, quinidin sulphate should be given a fair trial as it has proved successful in restoring sinus rhythm in a few such cases. If, on the other hand, auricular flutter develops during quinidin therapy in cases of auricular fibrillation, it is wise to stop the drug and resort to digitalization although some observers have succeeded in producing a normal rhythm with further quinidin therapy.

To summarize then, quinidin has not fulfilled its early promise of becoming one of the more important drugs in the treatment of cardiovascular diseases. It occasionally produces emboli with hemiplegia or pulmonary infarcts and sometimes is the cause of sudden death. It eliminates premature contractions (extrasystoles) in a comparatively small number of cases. Digitalis has proved more successful in preventing the various types of paroxysmal tachycardias, except in the cases of ventricular tachycardia. Quinidin is useful in auricular flutter, but this arhythmia is relatively rare, and finally it is only the very occasional case of auricular fibrillation in which quinidin therapy seems justifiable.

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CORRECTION

On reviewing my discussion on "Chronic Pericarditis" published in the April, 1933, number of *Modern Concepts of Cardiovascular Disease,* I find that my statement concerning Wenckebach's sign needs amplification. Originally Wenckebach described the abnormal (paradoxical) change of contour of the thorax and abdomen on inspiration when pericardial adhesions fasten the heart both to anterior chest wall and to diaphragm. Normally these two structures move in opposite directions on inspiration, that is the anterior chest wall rises and moves outward while the diaphragm descends, but when there are extensive pericardial adhesions the lower part of the anterior chest wall and upper abdomen tend to be pulled in by the diaphragmatic descent on inspiration, while the diaphragm itself is thus hampered in its movement. This paradoxical movement may be seen by inspection of the body from the side during deep respiration but it is best made out by fluoroscopy. If the adhesions are chiefly to the anterior chest wall and involve the diaphragm little or not at all then the heart as stated in my article on chronic pericarditis will rise with the sternum on inspiration instead of following the diaphragm down as it normally should do.

Also I should have added that with extensive adhesive pericarditis especially of the constrictive type (Pick's disease) low voltage in the electrocardiogram is a common finding and probably of greater significance than a failure of the electrical axis to shift with change of body position.

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SELECTED ABSTRACT

Rosenblum, H., Hahn, R. G., and Levine, S. A. Epinephrine: Its Effect on the Cardiac Mechanism in Experimental Hyperthyroidism and Hypothyroidism. Arch. Int. Med. 51:279: Feb. 1933.

Rosenblum and associates have shown that in rabbits with hyperthyroidism produced by feeding desiccated thyroid or injecting thyroxine, auricular fibrillation could be induced by injections of epinephrine which were insufficient to bring about this arrhythmia in normal or thyroidectomized (hypothyroid) rabbits. Similarly paroxysmal ventricular tachycardia occurred much more commonly after epinephrine in the former than in the latter group. The study lends support to the conception that the suprarenal glands may influence the occurrence of auricular fibrillation in hyperthyroidism. M.N.F.